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DATE: Tuesday, June 28, 2005

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	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR		
☐	L34	L33 and APO-3	34
☐	L33	L32 and apoptosis	172
☐	L32	l30 and antagonist	270
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☐	L30	(APO)same(antibod?)	952
☐	L29	L28 and apo-3	2
☐	L28	424/178.1.ccls.	1051
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☐	L21	L18 and APO-3	0
☐	L20	L18 and Apo-3	0
☐	L19	L18 and Apo-3 antibody	217182
☐	L18	435/7.21.ccls.	2559
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☐	L10	435/7.1.ccls.	10005
☐	L9	L8 and anti-Apo-3	9
☐	L8	L7 and APO-3	157
☐	L7	(ashkenazi)	2569
☐	L6	(Avi)adj(J)adj(ashkenazi)	0
☐	L5	(A)adj(ashkenzai)	0

☐	L4	(ashkenzai)same(APO-3)	0
	DB=USPT; PLUR=YES; OP=OR		
☐	L3	US-6469144-B1.did.	1
	DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR		
☐	L2	(APO-3)same(antibod?)	19
☐	L1	(APO-3)same(antibod?)same(antagonist)same(apoptosis)same(NF)adj (kappa)adj(B)adj(activation)	0

END OF SEARCH HISTORY

1: Science. 1989 Jul 21;245(4915):301-5.

Monoclonal antibody-mediated tumor regression by induction of apoptosis.

Trauth BC, Klas C, Peters AM, Matzku S, Moller P, Falk W, Debatin KM, Krammer PH.

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To characterize cell surface molecules involved in control of growth of malignant lymphocytes, monoclonal antibodies were raised against the human B lymphoblast cell line SKW6.4. One monoclonal antibody, anti-APO-1, reacted with a 52-kilodalton antigen (APO-1) on a set of activated human lymphocytes, on malignant human lymphocyte lines, and on some patient-derived leukemic cells. Nanogram quantities of anti-APO-1 completely blocked proliferation of cells bearing APO-1 in vitro in a manner characteristic of a process called programmed cell death or apoptosis. Cell death was preceded by changes in cell morphology and fragmentation of DNA. This process was distinct from antibody- and complement-dependent cell lysis and was mediated by the antibody alone. A single intravenous injection of anti-APO-1 into nu/nu mice carrying a xenotransplant of a human B cell tumor induced regression of this tumor within a few days. Histological thin sections of the regressing tumor showed that anti-APO-1 was able to induce apoptosis in vivo. Thus, induction of apoptosis as a consequence of a signal mediated through cell surface molecules like APO-1 may be a useful therapeutic approach in treatment of malignancy.

PMID: 2787530 [PubMed - indexed for MEDLINE]

1: Curr Biol. 1996 Dec 1;6(12):1669-76.

Apo-3, a new member of the tumor necrosis factor receptor family, contains a death domain and activates apoptosis and NF-kappa B.

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BACKGROUND: Two receptors that contain the so-called "death domain' have been described to date: tumor necrosis factor receptor 1 (TNFR1) and Fas/Apo-1 (CD95); both belong to the TNFR gene family. The death domain of TNFR1 mediates the activation of programmed cell death (apoptosis) and of the transcription factor NF-kappa B, whereas the death domain of CD95 only appears to activate apoptosis. RESULTS: We have identified an additional member of the TNFR family, which we have named Apo-3. Apo-3 is a transmembrane protein of approximately 47 kDa that has similarity of members of the TNFR family in its extracellular, cysteine-rich domains. In addition, Apo-3 resembles TNFR1 and CD95 in that it contains a cytoplasmic death domain. The Apo-3 gene mapped to human chromosome 1p36.3, and Apo-3 mRNA was detected in several human tissues, including spleen, thymus, peripheral blood lymphocytes, small intestine and colon. Ectopic expression of Apo-3 in HEK293 or HeLa cells induced marked apoptosis. CrmA, a poxvirus inhibitor of Ced-3-like proteases which blocks death signaling by TNFR1 and CD95, inhibited Apo-3-induced apoptosis. Ectopic expression of Apo-3 also induced the activation of NF-kappa B. Apo-3 did not specifically bind to the Apo-2 ligand, suggesting the existence of a distinct ligand for Apo-3. CONCLUSIONS: These results identify Apo-3 as a third member of the TNFR family that activates apoptosis, and suggest that Apo-3, TNFR1 and CD95 engage a common apoptotic cell-death machinery. Apo-3 resembles TNFR1 because it can stimulate NF-kappa B activity and regulate apoptosis. Apo-3 mRNA is expressed in various tissues, consistent with the possibility that this receptor may regulate multiple signaling functions.